CERTIFICATE OF MAILING BY "EXPRESS MAIL"

Express Mail Label No.: EL 984097703 US Date of Deposit: March 26, 2004

I hereby certify that this paper or fee is being deposited with the United States Postal Service "Express Mail Post Office to Addressee" service under 37 C.F.R. § 1.10 on the date indicated above and is addressed to: MS Non-Fee Amendment, Commissioner for Patents, P.O. Box 1450, Alexandria, VA 22313-1450.

(Marian Christopher)

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

In the application of:

MILLER, et al

Serial No.: 08/487,312

Filing Date: June 7, 1995

For: BOVINE GROWTH HORMONE

Examiner: C. Saoud

Group Art Unit: 1646

Declaration of Robert B. Petersen, Ph.D.
Pursuant to 37 C.F.R. § 1.132

MS Non-Fee Amendment
Commissioner for Patents
P.O. Box 1450
Alexandria, VA 22313-1450

Dear Sir:

I, Robert B. Petersen, Ph.D. declare as follows:

1. I am an Associate Professor of Pathology and Neuroscience at Case Western Reserve University.

2. I have been conducting research in the field of prion disease for 12 years. My research expertise encompasses the fields of cell and molecular biology of prion disease. My education, awards and honors, professional society memberships, relevant training and work experience, publications,

published abstracts and invited lectures and committee memberships are set forth in the attached Curriculum Vitae.

3. I have worked with recombinant models for the past 19 years in which proteins were expressed and analyzed.

4. The objective of this declaration is to evaluate the risks of pituitary derived versus recombinant derived bovine growth hormone for the development of Mad Cow disease.

5. Prior to preparing this declaration, I reviewed and became familiar with the following: (1) U.S. Patent No. 3,265,579 (hereinafter "Daniel's"); (2) U.S. Patent Application No. 487, 312 (hereinafter the "312 Application"); (3) the opinion of the U.S. Board of Patent Appeals and Interferences, Appeal No. 1998-1851 (March 26, 2003); and (4) Prusiner, Proc Natl Acad Sci USA **95**: 13363.

6. I have concluded that recombinant bovine growth hormone poses essentially no risk of causing Mad Cow disease whereas the pituitary derived hormone has a high risk, if Mad Cow disease is present in the population.

Bovine Growth Hormone

7. Bovine growth hormone, BGH, is a bovine derived protein comprised of 191 amino acids. BGH is produced in the pituitary, which is located deep in the brain near the brainstem. Since hormones are produced in the body in vanishingly small quantities, BGH is purified from the pooled pituitary glands from many cattle. These pools are typically on the order of greater than1,000 pituitary glands.

8. A recombinant form of BGH is used in agriculture in the US to promote growth and milk production in cows. The recombinant BGH is administered to cows by subcutaneous injection

[http://www.monsantodairy.com/about/benefits/att3.html; http://www.fda.gov/cvm/efoi/section2/140872.pdf]. The recombinant form is produced using DNA vectors transformed into unicellular organisms.

BSE

9. Bovine spongiform encephalopathy, BSE, or "Mad Cow" is a disease of cattle that was recognized in the United Kingdom in the mid-1980s. The disease was found to be a prion disease. Prions are a novel class of infectious agents lacking a nucleic acid genome and comprised primarily, if not only, of protein. The prion protein, which is a normal cellular protein, is believed to undergo a change in conformation rendering it pathogenic.

10. BSE is believed to have arisen from the ingestion of contaminated feed derived from scrapie-infected sheep. Scrapie was the first prion disease recognized and affects sheep and goats. Once cattle became infected, a cycle was formed due to the practice of using rendered cattle in feed. From cattle, the disease spread to the London zoo, domestic cats, and, in 1996, the first recognized case of the human disease variant Creutzfeldt-Jakob disease (vCJD).

11. While the UK, Europe and Japan currently test cattle for BSE only limited testing is done in the US. Testing is both difficult and expensive, taking at least a day to perform. These tests were not available prior to the late 1990s. In fact, the first case of BSE was not noted until around 1985. The tests currently in use the brainstem as the test substrate since it contains uniformly high amounts of the prion. The gold standard test is the bioassay in which brain homogenate is injected into the brain of a susceptible animal. However, this test is not practical for routine screening since the latency period before disease development is in excess of 60 days.

12. BSE has the potential to severely impact the US economy as evidenced by the regulation of its possession, use or transfer (9CFR121.3) promulgated under the Public Health Security and Bioterrorism Preparedness and Response Act of 2002 (PL 107-188). In the years following the outbreak of BSE, the UK is estimated to have lost approximately $3 billion to the ban of importation of beef from the UK. Due to the difference in size of the US cattle industry, the impact of BSE on the US would be greater by at least an order of magnitude.

13. Given the economic and safety risks BSE presents, regulatory agencies should not allow the use of pituitary derived BGH in cows, especially given the availability of the safe recombinant form.

CJD

14. Creutzfeldt-Jakob disease, CJD, is a progressive neurological disease of humans that occurs sporadically, by inheritance, or by infection. It is also believed to be caused by prions. Both CJD and vCJD are invariably fatal and diagnosis is only possible post-mortem.

15. Infection of humans with CJD has come about by the use of contaminated corneas and dura mater grafts, surgical instruments, growth hormone and contaminated beef. The last cause results in variant CJD, vCJD, that has been found in individuals decades younger than the age typically associated with CJD, 28 years old versus 55-60 years of age. The correlation of vCJD with BSE resulted in plummeting demand for beef in the UK and Europe. For example, after the discovery of BSE in Italy demand for beef dropped 70% overnight.

16. CJD has also been caused in humans who received human growth hormone derived from pools of human pituitaries. Human growth hormone has been used to treat some forms of growth disorders since the 1960s. In the 1970s in the US and the UK use of pituitary derived growth

hormone contaminated by prions resulted in CJD in the recipients. This unfortunate occurrence was repeated in France the mid-1980s where two contaminated lots of growth hormone were produced. Over 200 of the children treated with the contaminated growth hormone developed CJD.

17. These cases of iatrogenic CJD prompted the adoption in the US and Europe of using recombinant human growth hormone for treatment (rather than the previously used pituitary derived form) since recombinant production precludes the co-purification of the prion disease agent, and consequently has no risk of transmitting CJD. (This is discussed in more detail, below.)

BSE Risk Associated with Pituitary Derived BGH

18. There is a risk of BSE associated with the use in cattle of pituitary derived growth hormone.

19. Central nervous system tissue, including the brainstem, is a source of infectious prions that cause BSE.

20. The location of the pituitary gland makes it susceptible to contamination from infectious agents found in the brain and particularly from the brainstem. It is difficult to extract the pituitary from the brain without contamination. This is compounded by the pooling of thousands of pituitary glands to isolate useable amounts of growth hormone.

21. If pituitaries in the pool used to purify the pituitary derived form of BGH were infected, then standard purification techniques (like those described in Daniels) would not remove the BSE agent. (In other words, the Daniels method would co-purify the BSE agent if it was present in the pituitary starting material.)

22. Moreover, the method of administering cows with BGH (inoculation) would allow transmission of BSE if the hormone used was contaminated, as occurred in the human cases (see above).

23. The risk with pituitary derived BGH is enhanced by the fact that (a) testing cows for BSE, or testing samples of the BGH, is difficult and not routinely done in the US; (b) many pituitaries are needed to get enough hormone for use; (c) if a cow were infected, it would be difficult to extract pituitary without contamination from brain/brainstem resulting in contaminated BGH.

Recombinant BGH

24. The '312 Application describes the production of bovine growth hormone through recombinant technology. That is, it describes the production of the hormone by transforming an organism with the gene that encodes BGH and causing the organism to express relatively large quantities of the hormone.

25. The prion protein, which is the major if not only constituent of prions, has only been found in mammals.

26. Recombinant proteins are generally produced by heterologous expression in bacteria. The switch to a bacterially produced recombinant hormone would prevent the possibility of co-purifying a prion since the bacteria is incapable of making a prion.

27. In addition, the reagents used in the production of the recombinant hormone are also without risk for providing contaminating prions making the purified recombinant growth hormone essentially risk free.

28. The use of a recombinant protein source results in an easier purification and a product of higher purity than the form of hormone purified from pituitary tissue. The ease of purification is the result of all cells in the sample expressing high levels of the recombinant protein whereas in the tissue only some cells express and that at low levels.

29. Additionally, the production of the BGH by recombinant methods yields only one isoform of BGH. In comparison, natural BGH is produced as four different allelic and splice variants yielding four BGH isoforms. In short, pituitary derived BGH will consist of multiple isoforms whereas the recombinant BGH will consist of only one isoform.

Declaration

30. I hereby declare that all statements made herein of my own knowledge are true and all statements made on information and belief are believed to be true and further these statements were made with the knowledge that willful false statements and the like are punishable by fine or imprisonment or both under Section 1001 of Title 18 of the United States Code, and such willful false statements may jeopardize the validity of the application, any patent issuing thereon, or any patent to which this verified statement is directed.

Date: 3/24/04 Signed: _____

Robert B. Petersen, Ph.D.

March 26, 2004

CURRICULUM VITAE

Robert B. Petersen, Ph.D.
Associate Professor

Personal Information:

Social Security No: 388-66-0321
Office Address: Department of Pathology
Institute of Pathology
Case Western Reserve University
2085 Adelbert Road
Cleveland, Ohio 44106
216-368-6709
Fax:360-838-9226; e-mail: rbp@po.cwru.edu
Home Address: 32280 Creekside Drive
Pepper Pike, Ohio 44124
216-292-3589

Education:

1981 BS, Biochemistry, University of Minnesota, Graduated Magna Cum Laude

1985 Ph.D., Cell and Developmental Biology, University of Minnesota, NIH pre-doctoral trainee on Genetics training grant; American Cancer Society Institutional Grant recipient. Department of Genetics and Cell Biology, Advisor: Dr. Perry Hackett, 9/81 - 7/85

1986 Postdoctoral Research Fellow, University of Minnesota, Human Genetics Institute, Advisor: Dr. Perry Hackett, 8/85 - 10/86

1990 Postdoctoral Research Fellow, The University of Chicago, Awarded 3 year NIH National Research Service Award. The University of Chicago, Department of Molecular Genetics and Cell Biology, Advisor: Dr. Susan Lindquist, 11/86 - 1/90.

Faculty Appointment:

Instructor, The University of Chicago, Department of Molecular Genetics and Cell Biology, 2/90 - 8/91.

Assistant Professor, Department of Pathology, Case Western Reserve University, Cleveland, Ohio, 9/91 – 6/98.

Associate Professor, Department of Pathology, Case Western Reserve University, Cleveland, Ohio, 7/98 - present

Teaching Experience:

1992-98	Phase II, Nervous system, lecturer
1993	Path 516 - Experimental Pathology, lecturer
1997-	Path 444- Neurological Diseases: Pathological, Cellular, and Molecular Perspectives. Co-organized with MA Smith and lecturer
2002-	Anat 523, lecturer

Students advised:

1992	Tracy Craig, summer undergraduate research program
1992	Chris Erhardt, medical student
1992-93	David Shein, pathology fellow (co-sponsored with Dr. Mark Cohen)
1993-95, 97-99	Ishrat Zaidi, post-doctoral student
1996	Lorie Raetzman, rotating graduate student
1995-1998	Sabina Capellari, visiting resident
1997	Jeremy Sanford, rotating graduate student
1998-1999	Amy C. Long, graduate student

Residents advised

1996	Nancy Tresser, M.D., neuropathology fellow
1995	Rudolph Castellani, M.D., neuropathology fellow

Thesis Advisory Committee:

1996	Tobin Cheung, Pathology (S.G. Younkin advisor)
1998	Dacai Liu , Pathology (M. Sy advisor)
1998	Rosa Rivera, Biochemistry (outside reviewer)
1998	Xiang-Hong Song, Pathology (J. Teller advisor)
1998-	Krista Seanor, Pathology (D. Templeton advisor)
1999-	Tao Tao. Pathology (A. Tartakoff advisor)
1999-	Tong Liu, Pathology (M. Sy advisor)

Commitee chair for Dacai Liu and Xiang-Hong Song

Research Support (direct costs):

Active

Co-PI of entire project. National Institute of Health, Program Project, AG14359: Pathogenetic Mechanism of Prion Disease. $2,962,578 (6/97 - 5/02).

Principal Investigator Project 1(Program project AG14539): Transgenic models of prion diseases linked to the D178N mutation. $200,993 (6/99 - 5/00).

Past:

Co-Investigator. National Institute of Health, AG08155, Method to Extend Research in Time Award (MERIT), 04/01/94 - 03/31/99 (Pierluigi Gambetti, Primary applicant). $1,140,185.

Principal Investigator Project 3, Heat shock system: A model relevant to Alzheimer disease. National Institute of Health, Leadership and Excellence in Alzheimer Disease (LEAD) Award, 07/01/91 - 06/30/98 (Pierluigi Gambetti, Primary applicant). $464,181

Principal Investigator Pilot Project, Cellular and Molecular Pathology of Alzheimer Disease. National Institute of Health, Leadership and Excellence in Alzheimer Disease (LEAD) Award, 07/01/91 - 06/30/98 (Pierluigi Gambetti, Primary applicant). $45,557

Ad hoc Journal reviewer:

American Journal of Human Genetics

Ad hoc Journal reviewer (continued):

American Journal of Pathology
Biochemistry
BioTechniques
Brain Pathology
Journal of Biological Chemistry
The Lancet
Neurology
Journal of Neuroscience
Clinical Genetics
EMBO

Ad hoc Grant Reviewer:

Nebraska Experimental Program to Stimulate Competitive Research
1998 United States Department of Agriculture; Animal Health and Well-Being Program
1999 National Institute of Health, National Heart, Lung, and Blood Institute
Special Emphasis Panel, Development of Assay Methods for CJ Disease, RFA-NIH-HL-99-003.
2001 United States Department of Agriculture; Animal Genome

Professional Societies:

American Association for the Advancement of Science, 1981- present
American Society for Microbiology, 1984-present
American Society for Neuroscience, 1992- present
American Chemical Society, 1995-199
American Society for Cell Biology, 1997- present

Committees:

Department
Pathology Seminar Committee, 1993- present
Pathology Seminar Committee, Chair and Co-Director, 1994-98
Pathology representative to the BSTP Admissions Committee

School of Medicine

Member of the Alzheimer Center 1992- present
Judge: Irwin Lepow Medical Student Research Day, 1995.

Case Western Reserve University
Institutional Animal Care and Use Committee (IACUC)-1997- present
Institutional Animal Care and Use Committee (IACUC)- Chair 1998-2001.
Institutional Animal Care and Use Committee (IACUC)- Executive Committee- 1997-2001.
Judge: Michaelson - Morley Undergraduate Research, 1994, 1995.

Conference Committees:

Scientific Advisory Committee: Neurodegenerative Disorders: Common Molecular Mechanisms. Ocho Rios, Jamaica. February 23-28, 1997.

Scientific Advisory Committee: Neurodegenerative Disorders: Common Molecular Mechanisms. Ocho Rios, Jamaica. February 23-28, 1998.

Session Chair

Neurodegenerative Disorders: Common Molecular Mechanisms. Molecular Biology and Genetics. Ocho Rios, Jamaica. April 10-15, 1994.

Neurodegenerative Disorders: Common Molecular Mechanisms. Neuropathology: Clinical and Experimental Aspects. Ocho Rios, Jamaica. April 2-7, 1995.

Neurodegenerative Disorders: Common Molecular Mechanisms. Spongiform Encephalopathies: Experimental. Ocho Rios, Jamaica. February 23-28, 1997.

Neurodegenerative Disorders: Common Molecular Mechanisms. Prion Diseases. Ocho Rios, Jamaica. February 23-28, 1998.

Neurodegenerative Disorders: Common Molecular Mechanisms. Infectious Agents. Tobago, West Indies. April 8-14, 2000.

Consulting

Scientific Consultant- Efoora, Inc. (1/99-8/2000)
Chief Scientific Officer- Prion Developmental Laboratories, Inc. (9/2000-
Scientific Advisor, Genesis Bioventures, Inc. (4/01-11/02)
Director- Prion Developmental Laboratories, Inc. (1/2003-

Invited Seminars/ PlatformPresentations:

1. Petersen RB, McGarry TJ, DeBenedetti A, Golic KG, Lindquist S. Translational control during the heat shock response in *Drosophila. ASBC Fed Proc* **46**: 1958, 1987.

2. Petersen RB, McGarry TJ, Golic KG, Dellavalle RP, Lindquist S. Translational control during the heat shock response of *Drosophila*. *Regulation of Gene Expression by RNA Structure and Anti-messengers*, NATO/INSERM Workshop, Les Arcs, Savoie, France, February 1988.

3. Petersen RB and Lindquist S. Regulation of the *Drosophila* Hsp 70 gene. *Translation Control*, Cold Springs Harbor, September 1989.

4. Petersen RB, Vogel J, McGarry TJ, Lindquist S. Post-transcriptional regulation of the heat shock response in *Drosophila*. *Post-Transcriptional Control of Gene Expression.* NATO/EEC Workshop, Goslar, W. Germany, 1990.

5. Petersen RB, Goldfarb L, Tabaton M, Brown P, LeBlanc A, Montagna P, Cortelli P, Monari L, Autilio-Gambetti L, Gajdusek DC, Lugaresi E, Gambetti P. Fatal familial insomnia and one subtype of familial Creutzfeldt-Jakob disease: Effect of a polymorphism in the prion protein gene on a pathogenic mutation. *Neurology*, Vol 43, no. 4. *Am Acad Neurol 45th Annual Meeting*, April 1993.

6. Petersen RB, Tabaton M, LeBlanc A, Montagna P, Cortelli P, Monari L, Autilio-Gambetti L, Lugaresi E, Gambetti P. Fatal familial insomnia and the expanding family of prion diseases. *Transmissible and Non-Transmissible Neurodegeneration Disorders: Current Update*, Ocho Rios, Jamaica, 1993.

7. Petersen RB, Richardson SL, Chen SG, Parchi P, Urig CB, Gambetti P. The prion protein 178^{Asn} mutation alters processing in a transfected human cell line. *Society for Neuroscience*, Miami Beach, Florida, November 1994.

8. Petersen RB, Tresser NJ, Richardson SL, Gali M, Goren H, Gambetti P. A family with oculoleptomeningeal amyloidosis has a mutation in the transthyretin gene. *American Association of Neuropathologists*, San Antonio Texas, June 1995.

9. Petersen RB. Effect of PrP mutations on PrP metabolism. *Molecular Biology of Prions and Pathology of Prion Diseases.* Banbury Center CSH Laboratory, November 1995.

10. Petersen RB. FFI: genetic and molecular biology findings. *International Workshop on Fatal Familial Insomnia (FFI).* Bologna, Italy, May 1996.

11. Petersen RB. The inherited prion diseases and the effects of PrP mutations on the metabolism of PrP. *2nd Annual German American Frontiers of Science Symposium sponsored by the German American Academic Council in conjunction with the U.S. National Academy of Sciences, the Alexander Von Humboldt Foundation, the Max Planck Society and the German Academic Exchange*, June 1996

12. Petersen RB, Capellari S, Chen SG, Parchi P, Singh N, Zanusso G, Gambetti P. Effect of Pathogenic Mutations on Processing of the Prion Protein in Transfected Cells: Implications for Pathogenesis. *Neurodegenerative Disorders: Common Molecular Mechanisms.* Ocho Rios, Jamaica, 1997.

13. Petersen RB, Smith MA. Effect of Chronic Expression of Heme Oxygenase-1 in Neuronal Cells: Relevance to Neurodegenerative Diseases. *American Association of Neuropathologists*, Pittsburgh, Pennsylvania, June 1997.

14. Capellari S, Petersen RB. Studies of the post-translational modifications to the prion protein. *Neurodegenerative Disorders: Common Molecular Mechanisms*, Ocho Rios, Jamaica, 1998.

15. Petersen RB (lecturer). Workshop on "Prions and Aggregating Proteins." Atlanta, GA, June 1998.

16. Petersen RB (lecturer). Prions: From Protein to Pathogen. American Society of Clinical Pathologists Teleconference, June 1998.

17. Curagen Corp, New Haven, CT. August 1998.

18. Institute of Human Virology, University of Maryland. Baltimore, MD. November 1998.

19. Animal Resource Center, Case Western Reserve University, Continuing staff training. November 20, 1998.

20. Prion Disease: Current Health Issues. St. Joseph's Hospital, Milwaukee, WI. May 1999.

21. Prions: From Protein to Pathogen. Milwaukee Academy of Medicine. Milwaukee, WI. May 1999.

22. New Generation- New Approach/ Germany and the USA in an Age of Global History. Sponsored by the German -American Academic Council Foundation. Chicago, IL. November 1999.

23. Prion disease: an Update. Clinical Pathology Conference Series, CWRU, October, 2000.

24. Prion Disease: From Protein to Pathogen. Indiana University of Pennsylvania. Indiana, PA February 9, 2001.

25. Ruminations on the Origins and Impact of Mad Cow Disease. CWRU Women's Alumni Association, September 26, 2001.

26. Ruminations on the Biology of Prion Disease. Department of Neuroscience, CWRU, Cleveland, OH. January 16, 2002.

27. Diagnostics for TSEs: A Case for Simplicity, Keystone Symposium, Breckenridge, CO. April 5, 2003.

28. Prion Safety Training, Pathology P3 Staff, Institute of Pathology, Cleveland, OH. April 15, 2003.

29. Diagnostics for TSEs: A Case for Simplicity, University of Wyoming, Laramie, WY. April 18, 2003.

30. Cell Models of Inherited Human Prion Disease, Veterinary Laboratory Agency-Weybridge, New Haw, Addlestone, Surrey, United Kingdom. August 19, 2003.

31. Ethics of Using Animals in Research, Department of Pathology, CWRU, Sept. 12, 2003.

Publications:

1. Petersen RB, Hensel CH, Hackett PB. Identification of a ribosome-binding site for a leader peptide encoded by Rous sarcoma virus RNA. *J Virol* **51**: 722-729, 1984.

2. Petersen RB, Hackett PB. Characterization of ribosome binding on Rous sarcoma virus RNA *in vitro*. *J Virol* **56**: 683-690, 1985.

3. Hackett PB, Petersen RB, Albericio F, Gunderson SI, Hensel CH, Palmenberg AC, Barany G. Synthesis *in vitro* of a seven amino peptide encoded in the lead RNA of Rous sarcoma virus. *J Mol Biol* **190**: 45-57, 1986.

4. Petersen R, Lindquist S. The *Drosophila hsp70* message is rapidly degraded at normal temperature and stabilized by heat shock. *Gene* **72**: 161-168, 1988.

5. Hensel CH, Petersen RB, Hackett PR. Effects of alterations in the leader sequence of Rous sarcoma virus RNA on initiation of translation. *J Virol* **63**: 4986-4990, 1989.

6. Petersen RB, Moustakas A, Hackett PB. A mutation in the short 5'-proximal open reading frame on Rous sarcoma virus alters virus production. *J Virol* **63**; 4787-4796, 1989.

7. Petersen RB, Lindquist S. Regulation of HSP70 synthesis by messenger RNA degradation. *Cell Regulation* **1**: 135-149, 1989.

8. Yost HJ, Petersen RB, Lindquist S. RNA metabolism: strategies for regulation in the heat shock response. *Trends In Genetics* **6**: 233-227, 1990.

9. Lindquist S, Petersen RB. Selective translation and degradation of heat shock messenger RNAs in Drosophila. *Enzyme* **44**: 147-166, 1990.

10. Hackett RB, Dalton MW, Johnson DP, Petersen RB. Phylogenetic and physical analysis of the 5'leader RNA sequences of Avian retroviruses. *Nuc Acids Res* **19**: 6929-6934, 1991.

11. Petersen RB, Tabaton M, Berg L, Schrank B, Torack RM, Julien J, Vital C, Deleplanque B, Pendlebury WW, Drachman D, Smith TW, Davies P, Martin JJ, Oda M, Montagna P, Autilio-Gambetti L, Lugaresi E, Gambetti P. Analysis of the prion gene in thalamic dementia. *Neurology* **42**: 1859-1863, 1992.

12. Goldfarb LG, Petersen RB, Tabaton M, Brown P, LeBlanc AC, Montagna P, Cortelli P, Julien J, Vital C, Pendlebury WW, Haltia M, Willis PR, Hauw JJ, McKeever PE, Monari L, B Schrank, Swergold GD, Autilio-Gambetti L, Gajdusek C, Lugaresi E, Gambetti P. Fatal familial insomnia and familial creutzfeldt jakob disease: disease phenotype determined by a DNA polymorphism. *Science* **258**: 806-808, 1992.

13. Grant MP, Cohen M, Petersen R, Halmagyi GM, McDougall A, Tusa RJ, Leigh RJ. Abnormal eye movements in Creutzfeldt-Jakob disease. *Ann Neurol* **34**: 192-187, 1993.

14. Monari L, Chen SG, Brown P, Parchi P, Petersen RB, Mikol J, Gray F, Cortelli P, Montagna P, Ghetti B, Goldfarb LG, Gajdusek DC, Lugaresi E, Gambetti P, Autilio-Gambetti L. Fatal Familial Insomnia and Familial Creutzfeldt-Jakob disease: Different prion proteins determined by a DNA polymorphism. *Proc Natl Acad Sci USA* **91**: 2839-2842, 1994.

15. Smith MA, Kutty RK, Richey PL, Chader GJ, Wiggert B, Petersen RB, Perry G. Heme Oxygenase-1 is associated with the neurofibrillary pathology of Alzheimer Disease. *Am J Pathol* **145**: 42-47, 1994.

16. Yu J, Nagarajan S, Ueda E, Knez JJ, Petersen RB, Medof ME. Characterization of alternatively spliced PIG-A transcripts in normal and paroxysmal nocturnal hemoglobinuria cells. *Brazilian J Med Biol Res* **27**: 195-201, 1994.

17. Dellavalle RP, Petersen RB, Lindquist S. Preferential deadenylation of Hsp70 mRNA plays a key role in regulating Hsp70 expression in *Drosophilia melanogaster. Mol Cel Biol* **14**: 3646-3659, 1994.

18. Petersen RB, Tabaton M, Chen SG, Monari L, Richardson SL, Manetto V, Lanska DJ, Markesbery WR, Currier RD, Autilio-Gambetti, L, Gambetti P. Familial progressive subcortical gliosis: presence of prions and linkage to chromosome 17. *Neurology* **45**: 1062-1067, 1995.

19. Parchi P, Castellani R, Cortelli P, Montagna P, Chen SG, Petersen RB, Lugaresi E, Autilio-Gambetti L, Gambetti P. Regional distribution of protease resistant prion protein in Fatal Familial Insomnia. *Ann Neurol* **38**: 21-29, 1995.

20. Premkumar DRD, Smith MA, Richey PL, Petersen RB, Castellani R, Kutty RK, Wiggert B, Perry G, Kalaria RN. Induction of heme oxygenase-1 mRNA and protein in neocortex and cerebral vessels in Alzheimer's disease. *J Neurochem* **65**:1399-1402, 1995.

21. Tabaton M, Rolleri M, Masturzo P, Cammarata S, Angelini G, Hansen LA, Saitoh T, Petersen RB, Perry G, Richey P, Gambetti P, Bertolini S. Apolipoprotein E $\in$4 allele frequency is not increased in progressive supranuclear palsy. *Neurology* **45**: 1764-1765, 1995.

22. Petersen RB, Tresser NJ, Richardson SL, Gali M, Goren H, Gambetti P. A family with oculoleptomeningeal amyloidosis and dementia has a mutation in the transthyretin gene. *J Neuropathol Exp Neurol* **54**: 413, 1995.

22. Sforza E, Montagna P, Tinuper P, Cortelli P, Avoni P, Ferrillo F, Petersen R, Gambetti P, Lugaresi E. Sleep-wake cycle abnormalities in fatal familial insomnia. Evidence of the role of the thalamus in sleep regulation. *Electroencephalogr Clin Neurophysiol* **94**: 398-405, 1995.

23. Petersen RB, Parchi P, Richardson SL, Urig CB, Gambetti P. Effect of the D178N mutation and the codon 129 polymorphism on the metabolism of the prion protein. *J Biol Chem* **271**: 12661-12668, 1996.

24. Parchi P, Castellani R, Capellari S, Ghetti B, Young K, Chen SG, Farlow M, Dickson DW Sima AAF, Trojanowski JQ, Petersen RB, Gambetti P. Molecular basis of phenotypic variability in sporadic Creutzfeldt-Jakob disease. *Ann Neurol* **39**: 767-778, 1996.

25. Petersen RB, Goren H, Cohen M, Richardson SL, Tresser N, Lynn A, Gali M, Estes M, Gambetti, P. Transthyretin amyloidosis: A new mutation associated with dementia. *Ann Neurol* **41(3)**: 307-313, 1997.

26. Parchi P, Capellari S, Chen SG, Petersen RB, Gambetti P, Kopp N, Brown P, Kitamoto T, Tateishi J, Giese A, Kretschmar H. Typing prion isoforms. *Nature* **386**: 232-233, 1997.

27. Capellari S, Vital C, Parchi P, Petersen RB, Ferrer X, Jarnier D, Pegoraro E, Gambetti P, Julien J. Familial prion disease with a 144bp insertion in the prion protein gene in a Basque family. *Neurology* **49**: 131-141, 1997.

28. Raymond GJ, Hope J, Kocisko DA, Priola SA, Raymond LD, Bossers A, Ironside J, Will RG, Chen S, Petersen RB, Gambetti P, Rubenstein R, Smits MA, Lansbury PT, Caughey B. Molecular Assessment of the transmissibilities of BSE and scrapie to humans. *Nature* **388**: 285288, 1997.

29. Golic MM, Rong Y, Petersen R, Lindquist SL, Golic KG. FLP-mediated DNA mobilization to specific target sites in Drosophilia chromosomes. *Nuc Acids Res* **25**: 3665-3671, 1997.

30. Singh N, Zanusso G, Chen SG, Fujioka H, Richardson S, Gambetti P, Petersen RB. A cell model of inherited prion disease: prion protein aggregation reverted by low temperature. *J Cell Biol* **272**: 28461-28470, 1997.

31. Swietnicki W, Petersen RB, Gambetti P, Surewicz WK. pH-dependent stability and conformation of the recombinant human prion protein PrP (90-231). *J Biol Chem* **272**: 27517-27520, 1997.

32. Vital C, Gray F, Vital A, Parchi P, Capellari S, Petersen RB, Ferrer X, Jarnier D, Julien J, Gambetti P. Prion encephalopathy with insertion of octapeptide repeats: determines the type of cerebellar deposits. Neuropathol Appl Neurobiol **24**: 125-130, 1998.

33. Zanusso G, Liu D, Ferrari S, Hegyi I, Yin X, Aguzzi A, Hornemann S, Liemann S, Glockshuber R, Manson J, Brown P, Petersen RB, Gambetti P, Sy M-S. Prion protein expression: analysis with a panel of new monoclonal antibodies. PNAS USA **95**: 8812-8816, 1998.

34. Chapman J, Cervenáková L, Petersen RB, Lee HS, Estupinan J, Richardson S, Vnencak-Jones CL, Gajdusek DC, Korczyn AD, Brown P, Goldfarb L. APOE in non-Alzheimer amyloidoses: transmissible spongiform encephalopathies.
Neurology **51**: 548-53, 1998.

35. Swietnicki W, Petersen RB, Gambetti P, Surewicz. Familial mutations and the thermodynamic stability of the recombinant human prion protein. *J Biol Chem* **273**: 31048- 31052, 1998.

36. Siu IM, Robinson DR, Schwartz S, Kung HJ, Pretlow TG, Petersen RB, Pretlow TP. The identification of monoclonality in human aberrant crypt foci. *Cancer Res* **59**: 63-66, 1999.

37. Goedert M, Spillantini MG, Crowther RA, Chen SG, Parchi P, Tabaton M, Lanska DJ, Markesbery WR, Wilhelmson KC, Dickson DW, Petersen RB, Gambetti P. Tau gene mutation in familial progressive subcortical gliosis. *NatMed* **4:**.454-457, 1999.

38. Zanusso G, Petersen RB, Jin T, Kanoush R, Sharma Y, Ferrari S, Gambetti P, and Singh N. Proteosomal degradation and N-terminal protease-resistance of the codon 145 mutant prion protein. *J Biol Chem* **274:** 23396-23404, 1999.

39. Capellari S, Zaidi SIA, Urig CB, Perry G, Smith MA, and Petersen RB. Prion protein glycosylation: Sensitivity to Redox Change. *J Biol Chem* **274:** 34846-34850, 1999.

40. Takeda A, Perry G, Abraham NG, Dywer BE, Kutty RK, Laitinen JT, Petersen RB and Smith MA. Overexpression of heme oxygenase in neuronal cells, the possible interaction with Tau. *J. Biol. Chem.* **275**: 5395-5399, 2000.

41. Capellari S, Zaidi SIA, Long AC, Kwon EE, and Petersen RB.The Thr183Ala Mutation, Not the Loss of the First Glycosylation Site, Alters the Physical Properties of the Prion Protein. Journal of Alzheimer disease **2:** 27-35, 2000.

42. Gerst JL, Raina AK, Pirim, A McShea, A, Harris PLR, Siedlak SL, Takeda A, Petersen,RB, Smith MA. Altered Cell-Matrix Associated ADAM Proteins in Alzheimer Disease. J Neurosci Res **59**: 680-684, 2000.

43. Capellari S, Parchi P, Russo CM, Sanford J, Sy M-S, Gambetti P, Petersen RB. Effect of the E200K mutation on prion protein metabolism: Comparative study of a cell model and human brain. *Am. J Pathol* **157:** 613-622, 2000.

44. Kim HT, Russell RL, Raina AK, Harris PLR, Siedlak SL, Zhu X, Petersen RB, Shimohama S, Smith MA, and Perry G. Protein Disulfide Isomerase in Alzheimer Disease. *Antiox. Redox Signal.* **2**: 485-489, 2000.

45. Lu K, Wang W, Xie Z, Wong B-S, Li R, Petersen RB, Sy M-S, Chen SG. Expression and Structural Characterization of the Recombinant Human Doppel Protein. *Biochemistry* **39:** 13575-13583, 2000.

46. Wong B-S, Liu T, Li R, Pan T, Petersen RB, Smith MA, Gambetti P, Perry G, Manson JC, Brown DR, Sy M-S. Increased levels of oxidative stress markers detected in the brains of mice devoid of prion protein. *J. Neurochem.* **76:** 1-9, 2001.

47. Li, R., Liu, D., Zanusso, G., Liu, T., Fayen, J. D., Huang, J. H., Petersen, R. B., Gambetti, P., and Sy, M. S. The expression and potential function of cellular prion protein in human lymphocytes. *Cell. Immunol.* 207(1): 49-58, 2001.

48. Liu, T., Li, R., Wong, B. S., Liu, D., Tao, P., Petersen, R. B., Gambetti, P., and Sy, M.S.(2001). PrP^C is preferentially expressed on subpopulations of murine hematopoietic cells. *J. Immunology* 166: 3733-3742, 2001.

49. Liu, T., Zwingman, T., Li, R., Pan. T., Wong, B. S., Petersen. R.B., Herrup, K., Gambetti, P., and Sy, M. S. Differential expression of cellular prion protein in the mouse brain as detected with multiple anti-prion monoclonal antibodies. *Brain Research* 896:118-29, 2001.

50. Wong, B. S., Liu, T., Paisley, D., Li, R., Pan, T., Chen, S. G., Perry, G., Petersen, R. B., Smith, M. A., Melton, D, W., Gambetti, P., Brown, D. R., and Sy, M. S. (2001). Induction of HO-1 and NOS in Doppel-expressing mice devoid of PrP: Implications for Doppel function. *Mol. Cell. Neuro.* 17: 768-775, 2001.

51. Keisuke Hirai, Gjumrakch Aliev, Akihiko Nunomura, Hisashi Fujioka, Robert L. Russell, Craig S. Atwood, Anne B. Johnson, Yvonne Kress, Harry V. Vinters, Massimo Tabaton, Shun Shimohama, Adam D. Cash, Sandra L. Siedlak, Peggy L. R. Harris, Paul K. Jones, Robert B. Petersen, George Perry, and Mark A. Smith. Mitochondrial abnormalities in Alzheimer disease. *J Neurosci*, 21: 3017-3023, 2001.

52. Nunomura A, Perry G, Aliev G, Hirai K, Takeda A, Balraj EK, Jones PK, Ghanbari H, Wataya T, Shimohama S, Chiba S, Atwood CS, Petersen RB, Smith MA. Oxidative Damage is the Earliest Event in Alzheimer Disease. *J Neuropathol Exp Neurol,* 60: 759-767, 2001.

53. Tao Pan T,Monica Colucci,Boon-Seng Wong,Ruliang Li,Tong Liu, Robert B.Petersen,Shu Chen,Pierluigi Gambetti,and Man-Sun Sy. (2001). Novel differences between two human prion "strains "revealed by two-dimensional gel electrophoresis. *J. Biol. Chem.* 276: 37284-37288, 2001.

54. Dagvadorj, A, Petersen, R.B., Lee, H.S., Cervenakova, L, Shatunov, A, Budka, H, Brown, P, Gambetti, P, and Goldfarb, L.G. Spontaneous Mutations in the Prion Protein Gene Causing Transmissible Spongiform Encephalopathy. *Ann. Neurol.* 52: 355-359, 2002.

55. Tao Pan,Boon-Seng Wong,Tong Liu, Ruliang Li,Robert B. Petersen and Man-Sun Sy. Cell-surface prion protein interacts with glycosaminoglycans. *Biochem.J* **368**,81-90, 2002.

56. Capellari S, Parchi P, Campbell J, Posey DM, Petersen RB, and Gambetti P. Deletion of two repeats in the human prion protein gene: New polymorphism, disease causing mutation, or disease slowing factor? (in press).

57. Zaidi SIA, Capellari S, Richardson SL, Urig CB, Gambetti P, and Petersen RB. The F198S mutation in the prion protein alters glycosylation efficiency. (in preparation).

Book Chapters and Invited Articles:

1. Yost HJ, Petersen RB, Lindquist S. Post-transcriptional regulation of heat shock protein synthesis in Drosophila. In: *Stress Proteins in Biology and Medicine*, Cold Spring Harbor Press, Cold Spring Harbor, New York, pp. 379-409, 1990.

2. Gambetti P, Petersen R, Monari L, Tabaton M, Cortelli P, Montagna P, Lugaresi E, Autilio-Gambetti L. Fatal familial insomnia and the widening spectrum of prion diseases. In: *Spongiform Encephalopathies.* Allen I (ed). British Medical Bulletin, Vol 49, No. 4, pp. 980-994, 1993.

3. Gambetti P, Medori R, Manetto, V, Petersen R, LeBlanc A, Tritschler H.J., Monari L, Tabaton M., Autilio-Gambetti L. Fatal Familial Insomnia: A prion disease with distinctive histopathological and genotypic features. In: *Fatal Familial Insomnia: Inherited Prion Diseases, Sleep and the Thalamus.* Guilleminault C, Lugaresi P, Montagna P and Gambetti P (eds). Raven Press, 1994.

4. Petersen RB, Goldfarb L, Tabaton M, Brown P, Cortelli P, Montagna P, Autilio-Gambetti L, Gajdusek DC, Lugaresi E, Gambetti P. A novel mechanism of phenotypic heterogeneity demonstrated by the effect of a polymorphism on a pathogenic mutation in the PRNP (Prion Protein Gene). In: *Molecular Neurobiology*, Vol 8, pp. 99-103, 1994.

5. Gambetti P, Petersen R, Tabaton M, Gallassi R, Lugaresi E. Thalamic Dementia: Relationship with Fatal Familial Insomnia. *The Primary Degenerative Dementias Other than Alzheimer's Disease.* Clark A.W. (ed.), (in press).

6. Petersen RB, Richardson SL, Urig CB, Gambetti P. Analysis of the prion protein 178^{Asn} mutation linked to Fatal Familial Insomnia and Creutzfeldt-Jakob Disease in a transfected human cell line. In: *Research Advance in Alzheimer's Disease and Related Disorders.* Iqbal K, Mortimer J, Winblad B, Wisniewski H (eds). John Wiley & Sons, Ltd., Sussex, England, pp. 783-787, 1995.

7. Gambetti P, Parchi P, Petersen RB, Chen SG, Lugaresi E. Fatal Familial Insomnia and Familial Creutzfeldt-Jakob Disease: Clinical, Pathological and Molecular Features. *Brain Pathol* **5**: 43-51, 1995.

8. Parchi P, Petersen RB, Gambetti P. New Topics in familial prion diseases. *Seminars in Virology* 7: 181-187, 1996.

9. Petersen RB, Parchi P, Capellari S, Gambetti P. Fatal Familial Insomnia, Creutzfeldt-Jakob disease and the Prion Protein. *In: Protein Dysfunction in Human Genetic Disease.* Swallow D and Edwards Y (eds), BIOS Scientific Publishers, Oxford, England, pp. 243-254, 1997.

10. Parchi P, Capellari S, Zanusso G, Singh N, Gambetti P, Petersen RB. Inherited prion disease: Molecular pathology and Cell Models. *In: Prions: Molecular and Cellular Biology.* Harris DF (ed). Horizon Scientific Press, Wymondham, UK, pp. 63-81, 1998.

11. Parchi P, Petersen RB, Chen SG, Autilio-Gambetti L, Capellari S, Monari L, Cortelli P, Montagna P, Lugaresi E, Gambetti P. Molecular Pathology of Fatal Familial Insomnia. Brain Pathol 8: 539-548, 1998.

12. Gambetti P, Petersen RB, Parchi P, Chen SG, Capellari S, Goldfarb L, Gabizon R, Montagna P, Lugaresi E, Piccardo P, Ghetti B. Inherited prion diseases. Cold Spring Harbor Laboratory Press, Cold Spring Harbor, NY, pp. 509-583, 1999.

13. Petersen RB. Antemortem diagnosis of variant Creutzfeldt-Jakob disease. *Lancet* **353**: 163-164, 1999.

14. Wong B-S, Pan T, Liu T, Li R, Petersen RB, Jones IM, Gambetti P, Brown DR, Sy M-S. Prion Disease: A loss of Anti-oxidant Function? *BBRC* **275:** 249-252, 2000.

15. Takeda, A., Itoyama, Y., Kimpara, T., Kutty, R.K., Abraham, N.G., Dwyer, B.E., Petersen, R.B., Perry, G. and Smith, M.A. (2002) Role of heme catabolism in neurodegenerative diseases. In Heme Oxygenase in Biology and Medicine, Abraham, N.G. (Ed) and Alam, J. and Nath, K. (Assoc Eds). Kluwer Academic/Plenum Publishers, New York, pp 135-143.

16. Castellani, R.J., Atwood, C.S., Perry, G., Petersen, R.B. and Smith, M.A. (2002) Prion and Alzheimer diseases: the road to pathogenesis is paved with copper. In Prion Diseases and Copper Metabolism: BSE, Scrapie and CJD Research, Brown, D.R. (Ed.), Horwood Publishing, pp 96-101 and 195-250.

17. Perry, G., Taddeo, M.A., Petersen, R.B., Castellani, R.J., Harris, P.L.R., Siedlak, S.L., Cash, A.D., Liu, Q., Nunomura, A., Atwood, C.S., and Smith, M.A. (2003) Adventiously-bound redox active iron and copper are at the center of oxidative damage in Alzheimer disease. Biometals, 16, 77-81.

Abstracts:

1. Hensel CH, Petersen RB, Hackett PB. Mutations affecting inhibition of translation on Rous sarcoma virus mRNA. *Am Soc Microbiol*, Annual Meeting, 1985.

2. Hackett PB, Petersen RB, Hensel CH. Synthesis of a heptapeptide encoded in the leader RNA of Rous sarcoma virus may be important in the retrovirus life cycle. *RNA Tumor Viruses, CSH*, June, 1985.

3. Moustakas A, Petersen RB, Hensel CH, Hackett PB. Inactivation of an open reading frame of the Rous sarcoma virus leader RNA affects transformation of chicken embryo fibroblasts. *RNA Tumor Viruses CSH*, June, 1987.

4. Hackett PB, Hensel CH, Moustakas A, Petersen RB. Regulation of gene expression by a short open reading frame in the 5' leader sequence of Rous sarcoma virus RNA. *Int Meeting Biochem*, Beijing, China, August, 1987.

5. Hackett PB, Petersen RB, Moustakas A, Hensel CH. Translation of an open reading frame of the Rous sarcoma virus leader RNA affects transformation of chicken embryo fibroblasts. *Translational Control, CSH*, September, 1987.

6. Lindquist S, Borkovich K, Fortin M, Golic K, Petersen R, Rossi J, Susek R, Yost J. Studies on the function and regulation of hsps in yeast and fruit flies. *J Cell Biochem Suppl* (12 Part D), 251, 1988.

7. Petersen RB, Tabaton M, Medori R, Tritschler HJ, Berg L, Schrank B, Torack RM, Julien J, Vital C, Pendlebury W, Montagna P, Lugaresi E, Gambetti P. Familial thalamic dementia and fatal familial insomnia are prion diseases with the same mutation. *Third International Conference on Alzheimer's Disease and Related Disorders*, Abano Terme (Padova), Italy, July 12-17, 1992.

8. Petersen RB, Goldfarb L, Tabaton M, Brown P, LeBlanc A, Montagna P, Cortelli P, Monari L, Autilio-Gambetti L, Gajdusek DC, Lugaresi E, Gambetti P. Fatal familial insomnia and one subtype of familial Creutzfeldt-Jakob disease: Effect of a polymorphism on a pathogenic mutation in the prion protein. *FASEB J* 7: A627, 1993.

9. Tabaton M, Petersen RB, Monari L, Chen S, Lanska DJ, Markesbery WR, Currier RD, Manetto V, Autilio-Gambetti L, Gambetti P. Progressive subcortical gliosis: A new prion protein disease. *Society for Neuroscience*, Washington, D.C., November 7-12, 1993.

10. Monari L, Chen SG, Petersen R, Tabaton M, Gray F, Gambetti P, Autilio-Gambetti L. Differences in the protease-resistant prion protein fragments among human prion disease. *Society for Neuroscience*, Washington, D.C., November 7-12, 1993.

11. Petersen RB, Richardson SL, Urig CB, Autilio-Gambetti L, Gambetti P. Analysis of the prion protein 178^{Asn} mutation linked to Fatal Familial Insomnia and Creutzfeldt-Jakob disease in a transfected human cell line. *Fourth International Conference on Alzheimer's Disease and Related Disorders*, Minneapolis, Minnesota, July 29 - August 3, 1994.

12. Chen SG, Petersen R, Parchi P, Monari L, Wang W, Gambetti P, Gambetti L. Truncated forms of prion protein in human brain and cell lines. *Fourth International Conference on Alzheimer's Disease and Related Disorders*, Minneapolis, Minnesota, July 29 - August 3, 1994.

13. Chen SG, Monari L, Brown P, Parchi P, Petersen RB, Mikol J, Gray F, Cortelli P, Montagna P, Ghetti B, Goldfarb LG, Gajdusek DC, Lugaresi E, Gambetti P, Gambetti LA. Fatal familial insomnia and familial

Creutzfeldt-Jakob disease: Different prion proteins determined by a DNA polymorphism. *Fourth International Conference on Alzheimer's Disease and Related Disorders*, Minneapolis, Minnesota, July 29 - August 3, 1994.

14. Petersen RB, Richardson SL, Chen SG, Parchi P, Urig CB, Gambetti P. The prion protein 178^{Asn} mutation alters processing in a transfected human cell line. *Society for Neuroscience*, Miami Beach, Florida, November 13-18, 1994.

15. Chen SG, Parchi P, Petersen R, Monari L, Wang W, Gambetti P, Gambetti L. Truncated forms of prion protein in normal and pathological human brains. *Society for Neuroscience*, Miami Beach, Florida, November 13-18, 1994.

16. Smith, M.A., Richey, P.L., Kutty, R.K., Chader, G.J., Wiggert, B.,Petersen, R.B. and Perry, G. (1994) Heme oxygenase-1 in Alzheimerdisease pathology. Neurodegenerative Disorders: Common MolecularMechanisms, 22.

17. Petersen RB, Richardson SL, Urig CB, Parchi P, Zaidi SIA, Singh N, Chen SG, Gambetti P. Affect of mutations in the prion protein on processing in transfected human cell lines. Neurodegenerative Disorders: Common Molecular Mechanisms, Ocho Rios Jamaica 1995.

18. Petersen RB, Tresser NJ, Richardson SL, Gali M, Goren H, Gambetti P. A family with oculoleptomeningeal amyloidosis has a mutation in the transthyretin gene. *American Association of Neuropathologists*, San Antonio Texas, June 1995.

19. Smith MA, Premkumar DRD, Richey PL, Petersen RB, Castellani R, Kutty RK, Wiggert B, Perry G, Kalaria R. Induction of heme oxygenase-1 mRNA in neocortex in Alzheimer disease. Neurodegenerative Disease '95: Molecular and Cellular Mechanisms and Therapeutic Advances, 1995.

20. Singh N, Zanusso G, Gambetti P, Petersen RB. Cell culture models of prion diseases. *The American Society for Cell Biology*, 1995.

21. Petersen RB, Richardson SL, Urig CB, Parchi P, Singh N, Zaidi SIA, Capellari S, Gambetti P. Mutations in the prion protein alter processing in transfected human cells. *Society for Neuroscience*, San Diego, California, November 11-16, 1995.

22. Parchi P, Castellani R, Capellari S, Chen SG, Petersen RB, Young K, Farlow MR, Trojanowski JQ, Sima A, Ghetti B, Gambetti P. Molecular pathology of sporadic Creutzfeldt-Jakob disease: correlation with clinicopathological features and prion protein genotype. *Society for Neuroscience*, San Diego, California, November 11-16, 1995.

23. Zanusso GL, Singh N, Gambetti P, Petersen RB. In vitro study of prion protein processing in a subtype of GSS. *32nd Annual Meeting of the Italian Neuropathological Association,* Parma, Italy, June 3-5, 1996.

24. Petersen RB, Capellari S, Chen SG, Parchi P, Singh N, Zanusso G, Gambetti P. Inherited prion disease: a model system for studying protein processing. *6th International Congress on Cell Biology, 36th American Society for Cell Biology Annual Meeting,* San Francisco, California, December 7-11, 1996.

25. Feldman H, Green GJ, McGillivray BC, Berry K, Petersen RB, Gambetti P. Familial Insomnia: A Chinese kindred with the 178 Asparagine Prion protein gene mutation and 129 methionine homozygosity. *Canadian Congress of Neurological Sciences, 31st Annual Meeting,* London, Ontario, Canada, June 25-29, 1996.

26. Capellari S, Parchi P, Landis DD, Julien J, Vital C, Dickson DD, Richardson SL, Petersen RB, Gambetti P. Prion encephalopathy with octapeptide repeats insertion: a study of three families. *American Association of Neurology*, 1997.

27. Vital C, Gray F, Vital A, Parchi P, Capellari S, Petersen RB, Julien J, Gambetti P. Cerebellar modifications at immunohistochemical study (IHC) in 3 cases of prion encephalopathy with octapeptide repeat insertion (OPRI). *American Association of Neuropathologists*, Pittsburgh, Pennsylvania, June 1997.

28. Petersen RB, Smith MA. Effect of chronic expression of heme oxygenase-1 in neuronal cells: relevance to neurodegenerative diseases. *American Association of Neuropathologists*, Pittsburgh, Pennsylvania, June 1997.

29. Zanusso G, Petersen RB, Kanoush R, Gambetti P, Singh N. Mutant prion protein in Gerstmann-Sträussler-Scheinker disease Y145amber is degraded by the proteasomal pathway. *37th American Society for Cell Biology*, 1997.

30. Capellari S, Gambetti P, Petersen RB. Inter-relationship of the post-translational modifications to the prion protein. *37th American Society for Cell Biology*, 1997.

31. Lu, S.-Y., Smith, M.A., Abraham, N.G., Nishimura, R.N., Laitinen, J.T., Petersen, R.B. and Dwyer, B.E. (1997) H2O2 injury in neuroblastoma cells over- and under expressing heme oxygenase-1. Soc. Neurosci. Abst., 23, 2213.

32. Jin T, Zanusso G, Jing Y, Petersen RB, Gambetti P, Singh N. An abnormal isoform of the mutant prion protein in GSS Q217R is bound to the chaperone BiP. *Society for Neuroscience*, Los Angeles, California, November 7-12, 1998.

33. Perry G, Hirai K, Nunomura A, Petersen RB, Smith M. Neuronal mitochondrial proliferation in Alzheimer disease: Evidence for a fundamental metabolic abnormality. Mitochondria: Genetics, Health, and Disease. NIH Bethesda, MD. December 2, 1998.

34. Smith, M.A., Abraham, N., and Petersen, R.B. (1998) Stable overexpression of heme oxygenase-1 in neuronal cells results in cytoskeletal abnormalities and proliferative changes. *J. Neurochem.*, 70 (Suppl 1), S45.

35. Perry, G., Nunomura, A., Hirai, K., Petersen, R.B., and Smith, M.A. (1998) Is Alzheimer disease a metabolic disorder? Molecular and Genomic Effects of Tissue Oxygen Deprivation in Sleep Apnea, 6.

35. Pretlow TP, Siu I-M, Robinson DR, Schwartz S, Kung H-J, Petersen RB, Pretlow TG. Monoclonality in human aberrant crypt foci (ACF). AACR Annual Meeting, April 10-14, 1999.

36. Petersen RB, Capellari S, Zaidi SIA, Singh N, Gambetti P. Processing of the human prion protein. Keystone symposia April 10-16, 1999.

37. Takeda A., Petersen, RB, Smith MA. Effect of chronic expression of heme oxygenase -1 in neuronal cells: Possible interaction with Tau. *American Association of Neuropathologists*, Portland, Oregon, June 1999.

38. Jin T, Zanusso G, Jing Y, Petersen RB, Gambetti P, Singh N. Atypical fragments of the mutant prion protein in GSS Q217R, Y145Stop, and P102L are degraded by the proteosomal pathway and accumulate in distinct intracellular compartments. *Society for Neuroscience*, Miami Beach, Florida, October 1999.

39. Lee, H., Ogawa, O., Zhu, X., Castellani, R.J., Ghanbari, H., Petersen, R.B., Perry, G. and Smith, M.A. (2002) Aberrant expression of metabotropic glutamate receptor 2 (mGluR2) in the vulnerable neurons in Alzheimer's disease. Soc. Neurosci. Abstr., Program No. 785.16.

40. Zhu, X., Wang, Y., Ogawa, O., Lee, H.G., Raina, A.K., Fujioka, H., Shimohama, S., Atwood, C.S., Petersen, R.B., Perry, G. and Smith, M.A. (2002) Bcl-w plays a key neuroprotective role in Alzheimer's disease. J. Neurochem., 81 (Suppl 1), 76.

41. Smith, M.A., Takeda, A., Abraham, N.G., Dwyer, B.E., Kutty, R.K., Laitinen, J.T., Petersen, R.B. and Perry, G. (2002) Heme oxygenase and tau: synergistic antioxidants in neuronal cells. 2nd International Conference on Heme Oxygenase (HO/CO) and Cellular Stress Response, in press.

42. Lee, H.G., Ogawa, O., Zhu, X., O'Neill, M.J., Ghanbari, H., Petersen, R.B., Perry, G. and Smith, M.A. (2003) Aberrant expression of metabotrophic glutamate receptor 2 in the vulnerable neurons in Alzheimer disease. Research ShowCASE 2003, 70.